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INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)

(51) International Patent Classification 5 :
A61M 16/00, A62B 7/00, A61B 18/02, F16K 31/02

A1

(11) International Publication Number: **WO 94/22517**

(43) International Publication Date: 13 October 1994 (13.10.94)

(21) International Application Number: PCT/US94/03635

(22) International Filing Date: 1 April 1994 (01.04.94)

(30) Priority Data:
08/042,093 1 April 1993 (01.04.93) US

(60) Parent Application or Grant
(63) Related by Continuation
US 08/042,093 (CON)
Filed on 1 April 1993 (01.04.93)

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(81) Designated States: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KG, KP, KR, KZ, LK, LU, LV, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TT, UA, US, UZ, VN, European patent (AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE), OAPI patent (BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG).

Published
With international search report.
Before the expiration of the time limit for amending the claims and to be republished in the event of the receipt of amendments.

(54) Title: SLEEP APNEA CONTROL DEVICE

(57) Abstract

An apparatus for controlling obstructive sleep apnea comprises: a mask (12) for placement over a breathing orifice of a patient having obstructive sleep apnea; a hose (14) connected at a first end to said mask and at a second end to means for applying air pressure (14) to said mask and breathing orifice as said patient inhales and exhales; means for tracking (20) the inhalation and exhalation of said patient; and means (24) associated with said air pressure application means for triggering a burst of air pressure to said mask and orifice at a predetermined point of said inhalation or exhalation.

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SLEEP APNEA CONTROL DEVICE

Field of the Invention

The present is directed to methods and devices for controlling obstructive sleep apnea and the harmful effects of sleep apnea.

Background of the Invention

Obstructive Sleep Apnea (OSA) afflicts an estimated 1% to 3% of the general population and is due primarily to episodic upper airway obstruction during sleep. Those afflicted with sleep apnea experience sleep fragmentation and intermittent, complete or nearly complete cessation of breathing during sleep, with potentially severe degrees of oxyhemoglobin unsaturation. As a consequence, the apneic experiences severe interruption of sleep, and, as the disease progresses over periods of time, greater degrees of hypoxia. The duration of an apnea episode may exceed two minutes, resulting in the victim's arterial hemoglobin oxygen saturation falling below 50 percent.

The victim may be entirely unaware of the occurrence of these frequent obstructions to breathing. The symptoms of sleep apnea are generally excessive day-time sleepiness, and snoring. The nocturnal hypoxia may eventually lead to a number of further problems, such as cardiac arrhythmia, pulmonary hypertension, right heart failure, systemic hypertension, severe morning headache, intellectual and personality changes,

congestive heart failure and cognitive disfunction. Other effects of sleep apnea include right ventricular dysfunction with cor pulmonale, carbon dioxide retention during wakefulness as well as during sleep, and persistently reduced arterial oxygen tension. Sleep apneics may further have an elevated risk of accidents which involve driving and/or the operation of dangerous equipment.

Although the details of the pathogenesis of obstruction in sleep apnea patients have not been fully defined, it is generally accepted that the mechanism includes either anatomic or functional abnormalities of the upper airway which result in increased air flow resistance. It has also been hypothesized that a mechanism responsible for the known association between obesity and sleep apnea is excessive soft tissue in the anterior and lateral neck which applies sufficient pressure on internal structures to narrow the airway.

The treatment of sleep apnea has included such surgical interventions as uvulopalatopharyngoplasty, gastric surgery for obesity, and maxillo-facial reconstruction. Another mode of surgical intervention used in the treatment of sleep apnea is tracheostomy. These treatments constitute major undertakings with considerable risk of post-operative infection. Medical and psychosocial problems frequently interfere with the acceptance of tracheostomy, both by the patient and the physician, and this solution has generally been employed only in severe cases. Patients have frequently chosen to accept the discomfort associated with the disease rather than have a tracheostomy.

Pharmacologic therapy has in general also been disappointing, especially in patients with more than mild sleep apnea. In addition, there have been undesirable side effects from the pharmacologic agents used to treat sleep apnea.

Thus, medical practitioners have continued to seek non-intrusive modes of treatment for sleep apnea with high success rates and high patient compliance. These include, for example in cases caused to obesity, weight loss through exercise and regulated diet.

One of the most promising areas of work in the treatment of sleep apnea has included the use of continuous positive airway pressure (CPAP) to maintain the airway of the patient in a continuously open state during sleep. The continuous positive airway pressure applied in this manner has been found to provide a pneumatic "splint" for the nasopharyngeal airway U.S. Pat. No. 4,655,213 discloses sleep apnea treatments based on continuous positive airway pressure applied within the airway of the patient.

Continuous positive airway pressure (CPAP) has typically been applied to the patient, during periods of sleep, by way of the nose (Colin Sullivan et al., "Reversal of Obstructive Sleep Apnea by Continuous Positive Airway Pressure Applied Through the Nares", *The Lancet*, pp. 862-865 (Apr. 18, 1981)). Sullivan et al suggest that the application of low levels of pressure completely prevent upper airway occlusion during sleep, thus allowing the patient to have entire nights of uninterrupted sleep.

In the arrangement provided by Sullivan et al. two soft plastic tubes were shaped to fit snugly in each naris. The other ends of these tubes were inserted into a lightweight wide-bore tube, the arrangement being strapped to the patients face. A medical grade silicone rubber was then run over the nose and nares to provide a seal. Continuous positive pressure is produced by connecting one end of the wide-bored tube to an air compressor motor with variable speed control. The other end of the wide-bore tube was led away from the patient and narrowed, to provide a mechanical resistance. United States Letters Patent which are directed to CPAP and CPAP systems include U.S. Patent Nos. 5,146,918 to Kallok, et al.; 5,065,756 to Rapoport; and 4,919,128 to Kopala, et al.

Although CPAP has been found to be very effective and has been well accepted, it suffers from some of the same limitations as the surgery options. Initially, as shown in the above mentioned art, CPAP requires the patient to sleep with a cumbersome and uncomfortable facial mask in communication with an air compression system. A significant proportion of sleep

apnea patients do not tolerate CPAP well. Complications associated with CPAP therapy include nasal irritation, drying of the nasal mucosa, and patient discomfort.

The art has thus turned to modifications of the CPAP technique to minimize these side effect. For example, the prior art has turned to methods which provide a non-continuous or modulated flow of air to the patient. U.S. Patent No. 4,773,411, for example, discloses a method and apparatus for CPAP treatment which provides a substantially constant elevated airway pressure with periodic short term reductions of the elevated airway pressure to a pressure magnitude no less than ambient atmospheric pressure.

U.S. No. 5,148,802 to Sanders discloses the use of BiPAP or bi-level positive air pressure to increase user comfort. Sanders' system provides for the selective change of air pressure during either inhalation (IPAP) or exhalation (EPAP). Sanders'system utilizes a pressure control system to alter the flow of air both into and out of the mask.

While various systems have utilized modifications of CPAP to facilitate user comfort, none have been designed based upon an empirical model of the dimensional changes of the airway of the sleep apneic during inhalation and exhalation.

Experimental data suggest that the late phase of expiration may be the most vulnerable period for apneics, and may be the point where the collapse of the airway is most likely. Thus, pressure applied only in this phase of the cycle may be the most efficacious method in treating obstructive sleep apnea.

It would be desirable to develop an empirical method for determining the precise point at which a sleep apnea episode is most likely to occur. It would further be desirable to provide an apparatus and method for applying a pulse of positive air pressure to a patient at the point where the apnea episode is most likely to occur, based upon an empirical model.

Summary of the Invention

The present invention addresses these desires and needs. In accordance with the present invention, a method and apparatus for treating obstructive sleep apnea is disclosed. The apparatus and method of the present invention are based upon experimental data suggesting that the late phase of expiration may be the most vulnerable period for apneics, and may be the point where the collapse of the airway is most likely. Thus, pressure applied only in this phase of the cycle is believed to be the most efficacious method in treating obstructive sleep apnea. The data on which the present invention is based, provide detail of changes in upper airway area as a function of tidal volume during inspiration and expiration in patients with sleep apnea. The data were generated by means of computer tomographic imaging.

In a first embodiment, the present invention comprises an apparatus for controlling obstructive sleep apnea comprising: a mask for placement over a breathing orifice of a patient having obstructive sleep apnea; a hose connected at a first end to said mask and at a second end to means for applying air pressure to said mask and breathing orifice as said patient inhales and exhales; means for tracking the inhalation and exhalation of said patient; and means associated with said air pressure application means for triggering a burst of air pressure to said mask and orifice at a predetermined point of said inhalation or exhalation.

In a preferred method, the present invention is a method for controlling obstructive sleep apnea comprising the following steps: placing a mask for over a breathing orifice of a patient having obstructive sleep apnea; tracking the inhalation and exhalation of said patient; and applying a burst of air pressure to said mask and said breathing orifice at a predetermined point of said inhalation or exhalation.

Description of the Figures

Figure 1 is a schematic diagram of the changes in upper airway area as a function of tidal volume during the respiratory cycle.

Figure 2 is a block diagram a sleep apnea control system in accordance with the present invention.

Figure 3A is a comparison of the minimum airway size in inspiration in three subject groups.

Figure 3B is a comparison of the minimum airway size in expiration in the three subject groups.

Figure 4 is a graph of upper airway cross-sectional area plotted as a function of tidal volume in a normal subject over 4 anatomic levels.

Figure 5 is a graph of upper airway cross-sectional area plotted as a function of tidal volume in a snorer/mild apneic subject over 4 anatomic levels.

Figure 6 is a graph of upper airway cross-sectional area plotted as a function of tidal volume in an apneic patient over 4 anatomic levels.

Figure 7a is a graph comparison of upper airway area in early inspiration in the three subject groups.

Figure 7b is a graph comparison of upper airway area during the rest of inspiration in the three subject groups.

Figure 8a is a graph comparison of upper airway area at the beginning of expiration in the three subject groups.

Figure 8b is a graph comparison of the maximum area change in expiration (the difference in area between the maximum area in expiration and the last point in expiration) in the three subject groups

Figure 9 is a graph comparison of percent change ([maximum - minimum]) in airway area throughout inspiration and expiration in the three subject groups.

Figure 10 illustrates respiratory changes in upper airway dimensions for the three subject groups.

Detailed Description of the Preferred Embodiment

The present invention is described with reference to the enclosed Figures wherein the same numbers are utilized where applicable. The present invention is directed to a novel apparatus and method for controlling or treating sleep apnea.

Recent studies of the above name applicant have identified the changes that take place during a normal respiratory cycle in wakefulness in patients with sleep apnea. The changes in dimensions of the airway during the respiratory cycle are to be considered in four distinct phases.

Figure 1 illustrates schematic changes in upper airway area as a function of tidal volume in inspiration and expiration in patients with sleep apnea (OSA). In the first phase, identified as phase 1, there is an increase in upper airway area. The increase in airway area that occurs in apneics is greater than in normal individuals. This is compatible with recent demonstrations of increased activity of upper airway dilator muscles in patients with sleep apneas as compared to normal individuals.

In the second phase, shown as phase 2, corresponding to the remainder of the inspiration cycle, upper airway area is relatively constant. This is believed to be due to a balance established between the actions of negative intraluminal pressure, tending to collapse the airway, and that of upper airway musculature which tends to open the airway.

The third phase of the cycle, shown as phase 3 of Figure 1, is the beginning of expiration. At this point, the airway tends to expand.

Finally, towards the end of expiration, the airway narrows, as shown in phase 4 of Figure 1. The degree of narrowing is greater in patients with sleep apnea. It has been found that, in apneics, the airway heads towards a closed position before it is stopped by the onset of the next expiration, or phase 1 of Figure 1. The narrowing in the late part of expiration presumably is believed to be the result of a lack of positive intraluminal pressure, as in phase 3, or the activity of upper airway dilator muscles.

Example 1

The following is a discussion of a study which was performed by Applicants, and which forms the empirical basis for the methods and apparatus disclosed below.

The effects of respiration on upper airway caliber were studied using cine Computer Tomography (CT) in normal subjects, subjects with mild apnea, and subjects with obstructive sleep apnea. The nasal breathing of all three groups of subjects were scanned in the supine position. Eight millimeter thick axial slices were obtained at four anatomical levels from the nasopharynx to the retroglossal region every 0.4 seconds during the respiratory cycle. The major findings in the investigation included the following:

(1) that the upper airway was significantly smaller in apneics than normals, especially at the retropalatal low and retroglossal anatomical levels;

(2) in apneic patients, the airway had anterior-posterior configuration unlike the normal airway which had horizontal configuration with the major access in the lateral direction;

(3) in all three subject groups, little airway narrowing occurred in inspiration suggesting that the action of the upper airway dilator muscles balanced the effects of negative intraluminal pressure.

(4) in apneic patients, there was more enlargement of the airway in early inspiration, presumably reflecting increased upper airway muscle dilator activity;

(5) in expiration positive airway pressure resulted in expansion of the airway, this expansion being largest in apneics indicating that the apneic airway was more distensible than the normal airway;

(6) at the end of the expiration, the upper airway of apneics narrowed significantly. Thus, the airway in apneics moves towards a closed position at the end of expiration. The study data suggested that for treatment of obstructive sleep apnea, the most critical time in the respiratory cycle to

deliver positive airway pressure is towards the end of expiration.

The Study

All volunteers initially underwent one night of polysomnography using a NihonKohden Model EEG 4418A/K polygraph in a standard protocol. Standard parameters were monitored during the sleep studies including central occipital and frontal electro (EEG) right and left electro-oculograms (EOG), chin electromyogram (EMG), right and left anterior tibialis EMG, nasal and oral airflow, chest and abdominal wall motion, EKG, snoring with a microphone and oxyhemoglobin saturation oximeter.

A registered Polysomnographic Technologist scored the polysomnograms according to the criteria of Rechtschaffen and Kales using 30 second epochs. Based on the data, the subjects were divided into three groups on the basis of the polysomnography: (a) normal subjects having no history of snoring or sleep disorder breathing; (b) snore/mild apneic subjects; and (c) apneics.

An Imatron C-100 ultra fast scanner was used to perform cine CT studies. The subjects were studied in the supine position with their heads placed in a foam rubber head holder such as the CFI-409 Nuclear AP Head Immobilizer by X-ray Marketing Associated Inc. of Bensonville, Illinois. Thus, the subjects maintained a neutral position having their Frankford plane perpendicular to the floor. The subjects breathed exclusively through their noses and did not swallow during the CT scanning. Scans were obtained at four levels in the upper airway; at the nasopharynx, at two levels in the retropalatal region (high and low) and in the retroglossal anatomic levels. The cine CT provided for rapid acquisition of contiguous pairs of 8 mm thick slices corresponding to a given target ring encompassing the distance between the nasopharynx and retroglossal regions.

The subjects wore Downs CPAP Mask (Vital Signs Inc., Totowa, NJ) with a head strap during scanning. The one way valves in the two mask openings were removed. The opening of the mask adjacent to the mouth was occluded with a cork.

The nasal opening in the mask was connected with a short piece of tubing to a pneumotachograph (Yeager, S & M Instruments, Doylestown, PA), which was attached to a differential transducer (Validyne; Engineering Corporation, Northridge, California). The pneumotachograph was calibrated before each experiment with a one, two and a three liter syringe. A Datex CO_2 monitor (Puritan-Bennett Corporation, Los Angeles, California) was utilized to determine if a mask leak was present (elevated CO_2 monitor [Puritan-Bennett Corporation, Los Angeles, California]) was utilized to determine if a mask leak was present (elevated CO_2 around the perimeter of the mask). A Macintosh IIX computer, running on a commercially available laboratory control package called LabVIEW 2.0 (National Instruments, Austin, TX) digitize on-line (150 Hz) the flow signal from the subject.

The flow signal once digitized was integrated by the computer to obtain tidal volume. The computer also received the high voltage signal from the scanner indicating the time of acquisition of each slice. Therefore, tidal volume was synchronized with the time of each slice acquisition so that a correlation between scanning and physiologic data was obtained.

To quantify the cross-sectional area of each CT image of the upper airway, the edge between the soft tissue and the airway had to be objectively determined. An edge detection algorithm, was programmed into an image analysis software package called VIDA (volumetric image display analysis) to perform this function. The edge detection algorithm determined an edge or threshold objectively at each point around the airway. Using the algorithm, a polygon was then drawn manually on the computer screen estimating the edge of the airway. Each point around the circumference of this original polygon was adjusted in the computer algorithm in several discreet steps using a "half-maximum" criteria to determine an objective edge of the airway at that location and a new polygon was generated. The cross-sectional area of the final polygon was then quantified. This was done separately for images obtained at each anatomical level, and for each time point during the

respiratory cycle. For each image, anterior-posterior and lateral dimensions are determined by fitting the smallest possible bounding box to the polygon. The vertical dimension corresponded to the largest anterior-posterior length of the polygon and the "horizontal" dimension corresponded to the largest lateral length of the polygon.

For each image, the analysis was repeated three times and the average cross-sectional, anterior-posterior and lateral diameters of the upper airway were obtained. Using this methodology, airway geometrical changes were objectively determined. Once upper airway area was measured, it was plotted as a function of tidal volume to generate an area-tidal volume loop.

Distinct dynamical changes in airway size throughout the respiratory cycle were identified. Respiratory variation in upper airway area as a function of tidal volume of a represented normal subject, snorer/mild apneic and apneic patient, at all four anatomic levels are displayed in Figures 4-6 respectively. In all three subjects, the caliber of the airway remained relatively constant during inspiration. The upper airway and all four anatomic levels in each of the subjects enlarged in early expiration and narrowed towards the end of expiration.

The data set forth in Figures 4-6 indicate that in the normal and snorer\mild apneic subjects, upper airway cross-sectional area decreases slightly in the early stages of inspiration, enlarges towards the end of inspiration and is larger at end-inspiration than at end-expiration. In the apneic patient, the caliber of the upper airway remains relatively constant throughout most of inspiration. In the apneic patient shown in Figure 6, the airway enlarges from the end of expiration to the beginning of inspiration (see extrapolated line). To examine airway dimensions in early inspiration, the difference between the last point in expiration from the previous breath and the first point in inspiration of the breath displayed in Figures 4-6 are measured.

These data are presented graphical form in Figure 7A. (For each of the graphs, diagonal 3-D bar=normal subjects, white

3-D bar = snorer/mild apneic subjects, gray dotted 3-D bar = apneic patients). In the earliest part of inspiration, primarily at the nasopharynx and retropalatal high anatomic levels, the airway enlarges more in the apneic patients than in the normal or snorer/mild apneic patients. A detailed analysis of the data in all three subject groups supports these conclusions. In the normal and snorer/mild apneic subjects, upper airway caliber is significantly greater at the end of inspiration than at the beginning of inspiration at all four anatomic levels as shown in the graph of Figure 7B.

The upper airway was larger in expiration compared to inspiration in all three subject groups. Examination of the results for the representative subjects in Figures 4-6 indicated that upper airway area widens from the end of inspiration to the beginning of expiration and then narrows towards the end of expiration in all groups. In all subject groups, upper airway area at the first expiratory time point was significantly greater than at the end of inspiration at all four anatomic levels as shown in Figure 8A.

Later in expiration, the airway narrowed in all subject groups. To quantify the narrowing, the differences between the maximum area in expiration, and the area at the end of expiration were measured. These differences are displayed in the graph of Figure 8B. Within each subject group, these differences were statistically significant at all four anatomic levels. The apneic subjects showed significantly more airway narrowing at the latter part of expiration than the normal subjects at the retropalatal low and retroglossal anatomic levels. Thus, the upper airway enlarged from the end of inspiration to the beginning of expiration in all three subject groups. In the apneic group, there was significantly more airway enlargement at the beginning of expiration at the retropalatal low and retroglossal levels. The narrowing toward the end of expiration is significantly greater in the apnea group at the retropalatal low and retroglossal anatomic levels. Airway closure during an apneic event may be an expiratory rather than an inspiratory phenomenon.

It was next determined whether the changes in airway dimensions in the different subject groups were compatible with the hypothesis that the apneic airway is more "floppy". The medium percentage change in airway size (maximum minus minimum divided by minimum) throughout the respiratory cycle at each anatomic level was measured and are set forth at Figure 9. The apneic group had the largest overall change in airway size. Most of this percent change occurred during expiration.

The overall percent change in airway caliber during respiration was significantly greater in the apneic group when compared to the normal group. Significant differences between the apneic and normal groups in the percentage change in airway size during respiration was measured at all four anatomic levels.

Figure 10 represents the geometric configuration of the upper airway at four anatomic levels in the three subject groups drawn using the maximum anterior-posterior and lateral dimensions in expiration and the minimum anterior-posterior and lateral dimensions in inspiration. Figure 10 demonstrates that the apneic upper airway at all four anatomic levels has an anterior-posterior configuration unlike the normal airway which has a horizontal configuration. The snorer/mild apneic airway has an intermediate configuration between the normal and apneic upper airway. These data suggest that the apneic is compromised laterally altering the elliptical shape of the airway so that its longest diameter of the airway is no longer in the lateral direction but in the anterior-posterior direction.

Irrespective of anatomic level, the global minimum airway size is approximately two times smaller in apneics compared to normal subjects. In addition to upper airway narrowing in patients with sleep apnea, it was determined that the apneic airway has an anterior-posterior configuration unlike the horizontal configuration of the normal airway.

Both the increase in airway caliber from inspiration to expiration, as shown in Figure 8A, as well as the percent change in airway size during the inspiration and expiration, as shown in Figure 9 are larger in the apneics than the normals or

snorer/mild apneics (primarily at the retropalatal low and retroglossal levels). These data suggest that the apneic airway is more distensible (or "compliant") than the normal or snorer/mild apneic airway.

The results suggest that a more effective method of delivering positive airway pressure than CPAP or BiPAP is possible. The results show that the largest reduction in airway dimensions in patients with sleep apnea occurs at the end of expiration when the airway significantly narrows. The apneic upper airway behaves differently than the normal upper airway. The apneic airway is smaller and has a more anterior-posterior elliptical configuration than a normal airway. In early inspiration, the apneic airway enlarges more, presumably due to increased activity in the upper airway muscles. Subsequently, airway size remains relatively constant throughout the rest of inspiration suggesting that the effect of negative intraluminal pressure is counterbalanced by the action of the upper airway dilator muscles. In expiration, the apneic airway is more distensible than the normal airway. In later expiration, the apneic airway narrows more than in normals and appears to be headed towards a closed position prior to it enlarging at the onset of the next inspiration.

Therefore, the results suggest that positive airway pressure should be delivered near the end of expiration, and/or in early inspiration, to prevent the reduction in airway dimensions. It is at this phase of expiration, phase 4 of Figure 1, that the positive airway pressure should be applied as a pulse for a short duration to prevent the decline in airway dimensions that occur at this phase of the respiratory cycle. Alternatively, the pulse of pressure should be applied at the end of expiration and in early inspiration to prevent airway collapse. The results of the above discussed study suggest apparatus for controlling obstructive sleep apnea.

The study discussed herein further suggests an apparatus and method for controlling obstructive sleep apnea 10 as shown in Figure 2. In a simplest embodiment, the apparatus comprises a mask 12 for placement over a breathing orifice of a

patient having obstructive sleep apnea. A hose 14 is connected at a first end 16 to the mask 12 and at a second end to means 18 for applying air pressure to said mask and breathing orifice as said patient inhales and exhales. In a preferred embodiment, means 18 may comprise an air compressor. The apparatus will include means 20 for tracking the inhalation and exhalation of the patient. Means 20 may comprise an electronic monitor or mechanical sensor 22 which tracks the inhalation and exhalation of the patient. It is to be appreciated by those skilled in the art that sensor 22 may comprise any mechanical, electrical or electronic mechanism which can sense or track the inhalation or exhalation of the patient is applicable in the present invention. Finally, the embodiment includes means 24 associated with the air pressure application means 18 for triggering a burst of air pressure to the mask and breathing orifice of the patient a predetermined point of inhalation or exhalation. As noted above, in view of the above discussed experimental results, this predetermined point may be the end of the exhalation, the beginning of inhalation or other point pertinent to a particular patient.

The present invention has been described with reference to the enclosed Figures. It is to be appreciated that other embodiments will fill the spirit and scope of the present invention and that the true nature and scope of the present invention is to be determined in reference to the claims appended hereto.

CLAIMS

What is claimed is:

1. An apparatus for controlling obstructive sleep apnea comprising:

a mask for placement over a breathing orifice of a patient having obstructive sleep apnea;

a hose connected at a first end to said mask and at a second end to means for applying air pressure to said mask and breathing orifice as said patient inhales and exhales;

means for tracking the inhalation and exhalation of said patient; and

means associated with said air pressure application means for triggering a burst of air pressure to said mask and orifice at a predetermined point of said inhalation or exhalation.

2. The apparatus for controlling obstructive sleep apnea of claim 1 wherein said burst of air pressure is triggered toward the end of said exhalation.

3. The apparatus for controlling obstructive sleep apnea of claim 1 wherein said burst of air pressure is triggered at the beginning of said end inhalation.

4. An apparatus for controlling obstructive sleep apnea comprising:

a mask for placement over the nose of a patient having obstructive sleep apnea;

a hose connected at a first end to said mask and at a second end to air compressor means for applying air pressure to said mask and nose as said patient inhales and exhales;

means for tracking the inhalation and exhalation of said patient; and

means associated with said air pressure application means and said tracking means for triggering a burst of air pressure to said mask and orifice at a predetermined point of said inhalation or exhalation.

5. The apparatus for controlling obstructive sleep apnea of claim 4 wherein said burst of air pressure is triggered at the end of said exhalation.

6. The apparatus for controlling obstructive sleep apnea of claim 4 wherein said burst of air pressure is triggered at the beginning of said end inhalation.

7. A method for controlling obstructive sleep apnea of a patient having obstructive sleep apnea comprising the following steps:

tracking the inhalation and exhalation of the patient having obstructive sleep apnea; and

applying a burst of air pressure into the breathing orifice of said patient at a predetermined point of said inhalation or exhalation so as to prevent the collapse of said patient's airway.

8. The method for controlling obstructive sleep apnea of claim 7 wherein said burst of air pressure is applied at the end of exhalation.

9. The method for controlling obstructive sleep apnea of claim 7 wherein said burst of air pressure is applied at the beginning of inhalation.

10. A method for controlling obstructive sleep apnea comprising the following steps:

placing a mask for over a breathing orifice of a patient having obstructive sleep apnea;

tracking the inhalation and exhalation of said patient; and

applying a burst of air pressure to said mask and said breathing orifice at a predetermined point of said inhalation or exhalation so as to prevent the collapse of said patient's airway.

11. The method for controlling obstructive sleep apnea of claim 10 wherein said burst of air pressure is applied at the end of exhalation.

12. The method for controlling obstructive sleep apnea of claim 10 wherein said burst of air pressure is applied at the beginning of inhalation.

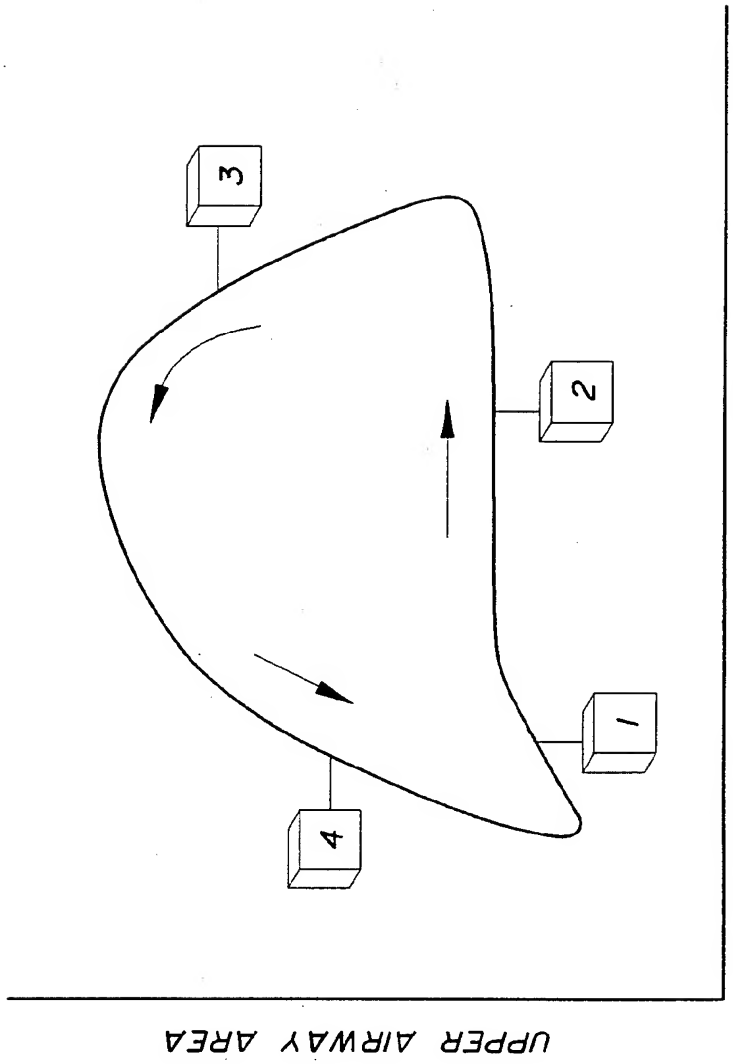

FIG. 1

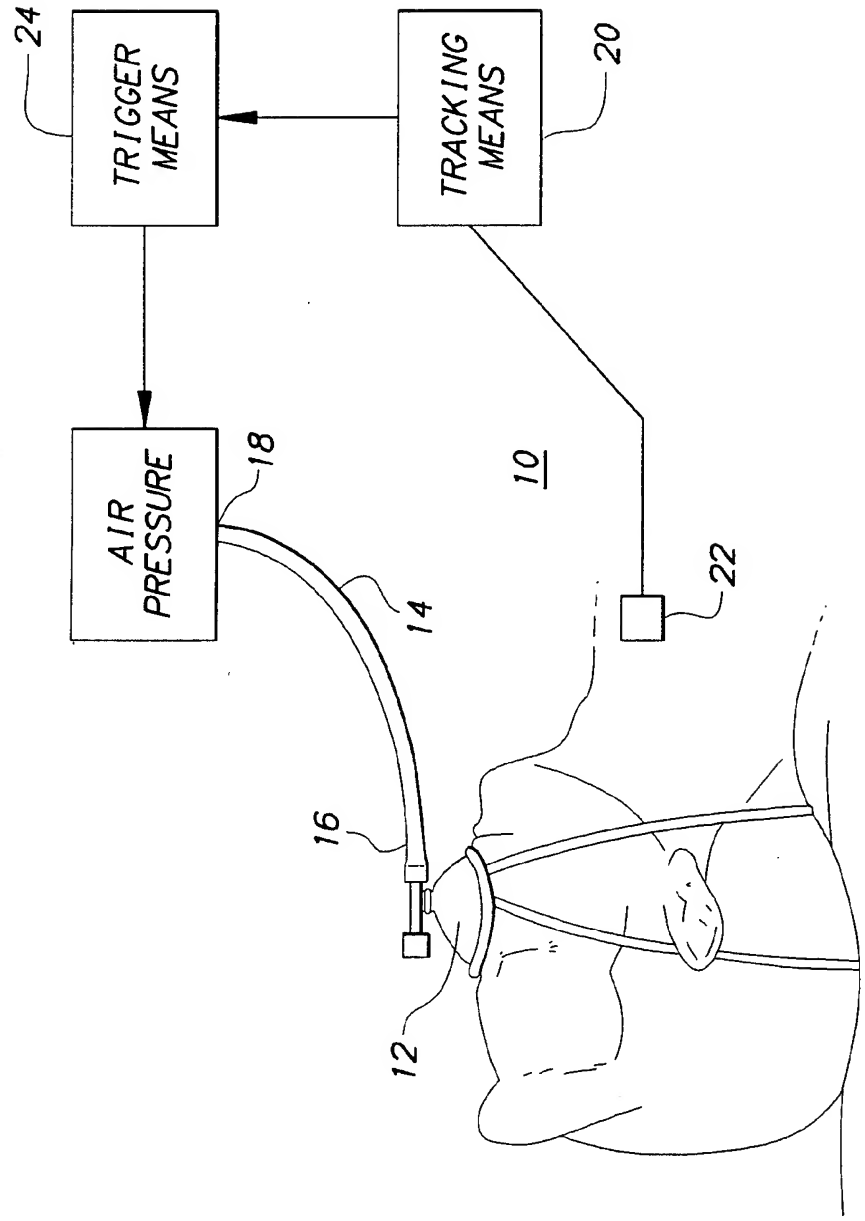
24
TRIGGER MEANS
TRACKING MEANS
20
AIR PRESSURE
18
10
14
22
16
12

FIG. 2

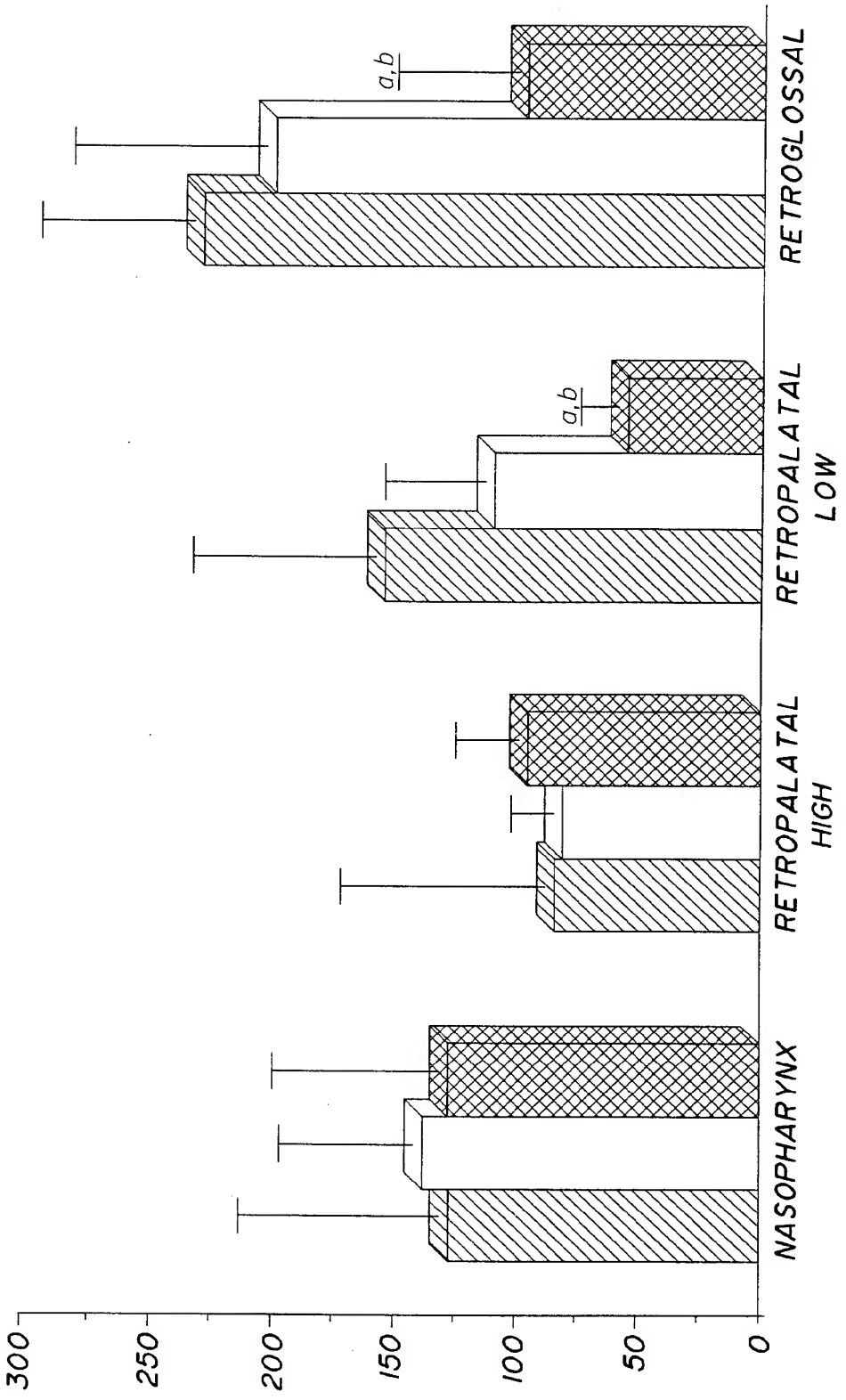
MINIMUM AREA IN EXPIRATION (mm^2)
300
250
200
150
100
50
0
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL
a,b
a,b

FIG. 3A

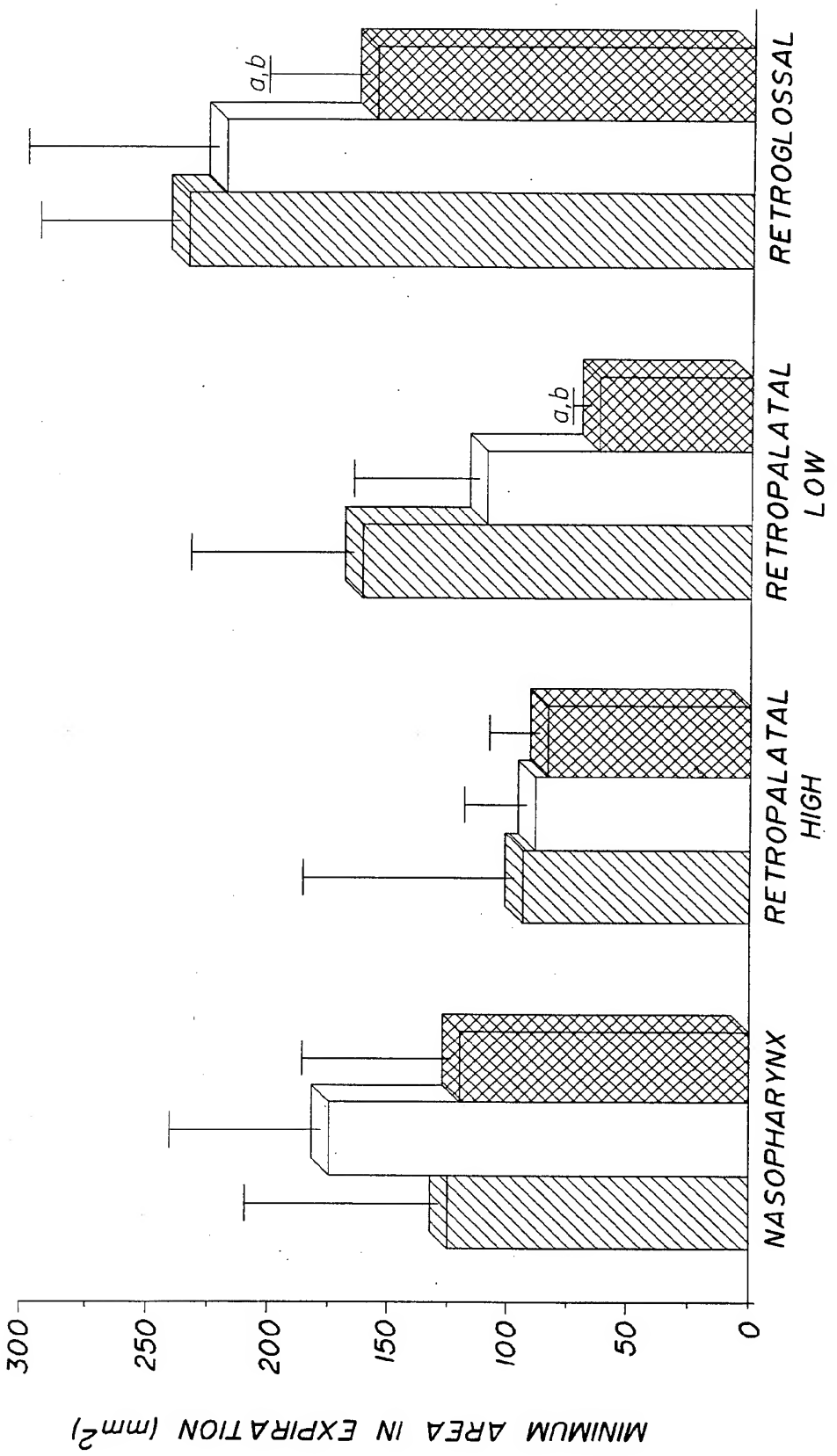
MINIMUM AREA IN EXPIRATION (mm^2)
300
250
200
150
100
50
0
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL
a,b
a,b

FIG. 3B

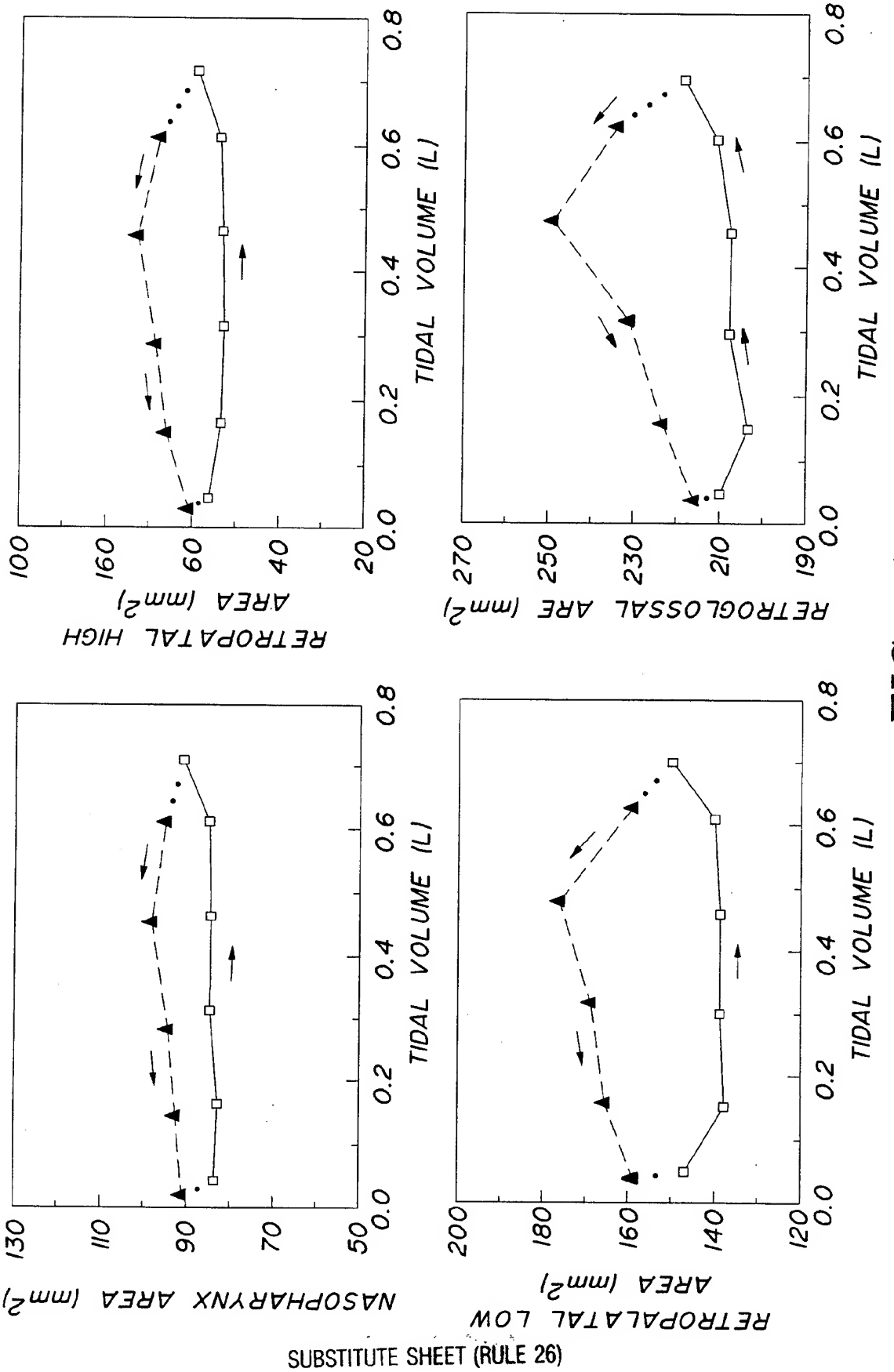
RETROPALATAL LOW
AREA (mm²)
200
180
160
140
120
0.0
0.2
0.4
0.6
0.8
TIDAL VOLUME (L)
RETROGLOSSAL ARE (mm²)
270
250
230
210
190
NASOPHARYNX AREA (mm²)
130
110
90
70
50
RETROPATAL HIGH
AREA (mm²)
100
160
60
40
20

FIG. 4

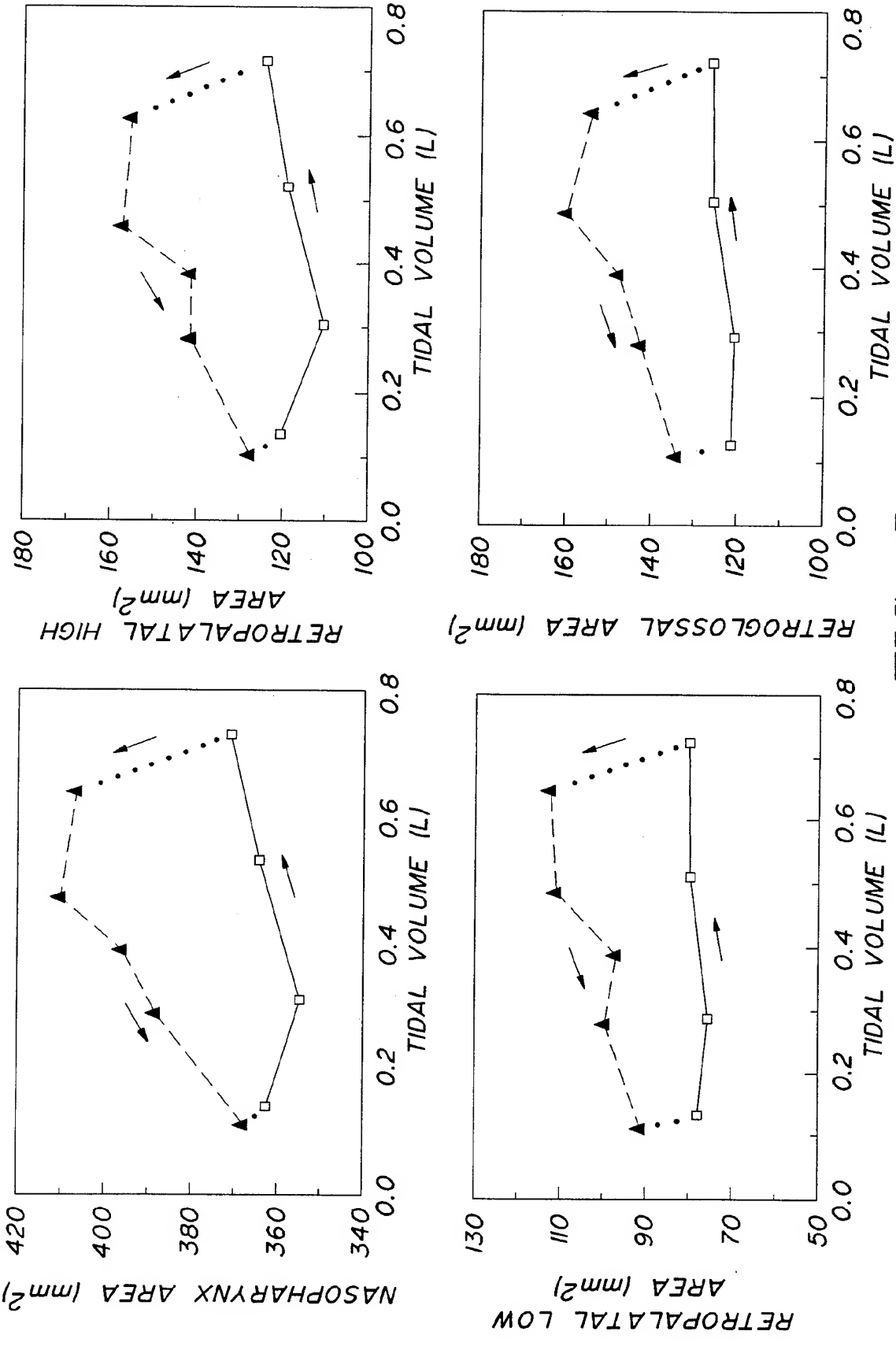
RETROPALATAL HIGH AREA (mm2)
RETROGLOSSAL AREA (mm2)
NASOPHARYNX AREA (mm2)
RETROPALATAL LOW AREA (mm2)
TIDAL VOLUME (L)
0.0
0.2
0.4
0.6
0.8
100
120
140
160
180
340
360
380
400
420
50
70
90
110
130

FIG. 5

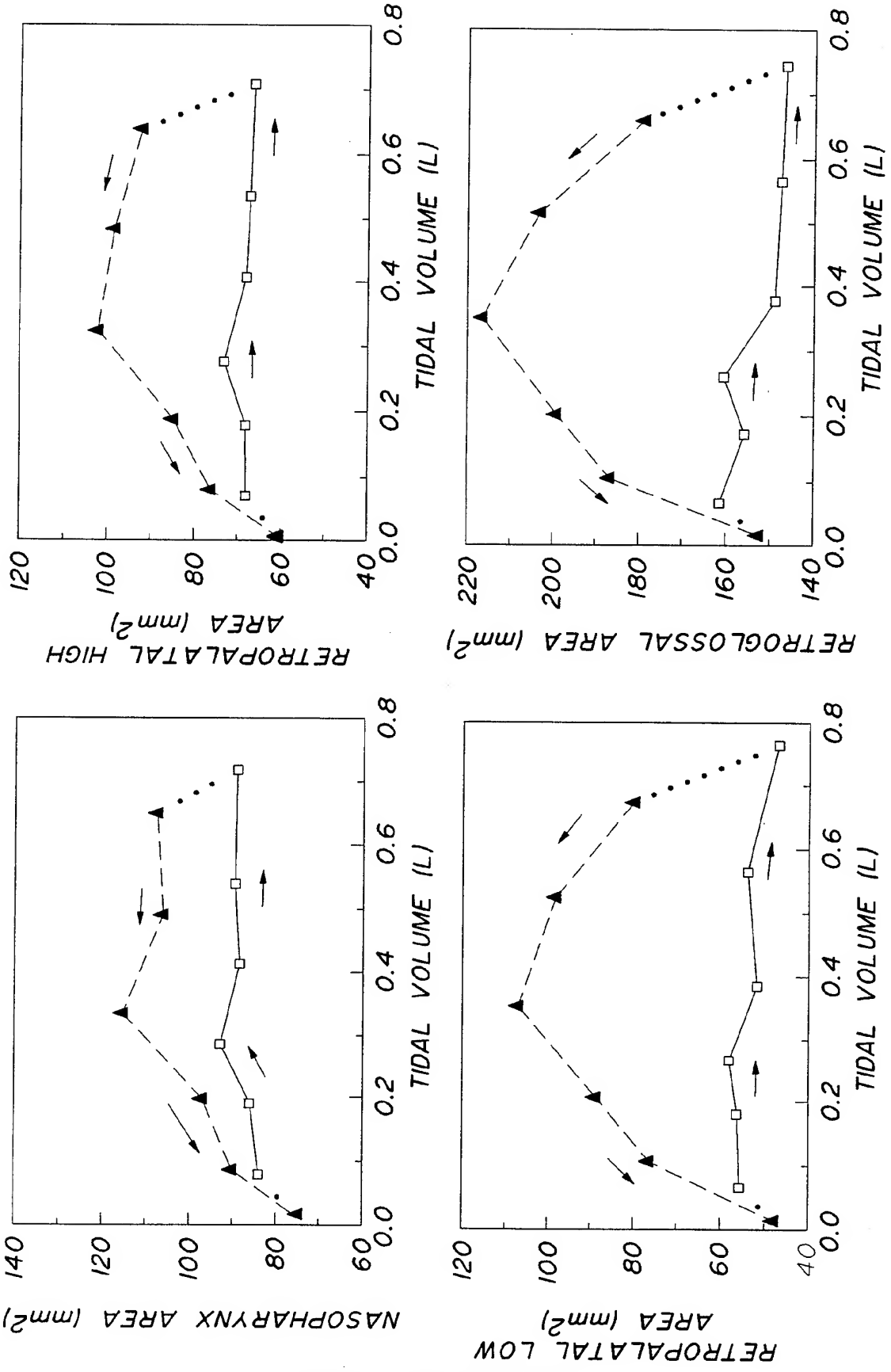
RETROPALATAL HIGH AREA (mm2)
RETROGLOSSAL AREA (mm2)
NASOPHARYNX AREA (mm2)
RETROPALATAL LOW AREA (mm2)
TIDAL VOLUME (L)
0.0
0.2
0.4
0.6
0.8
40
60
80
100
120
140
160
180
200
220

FIG. 6

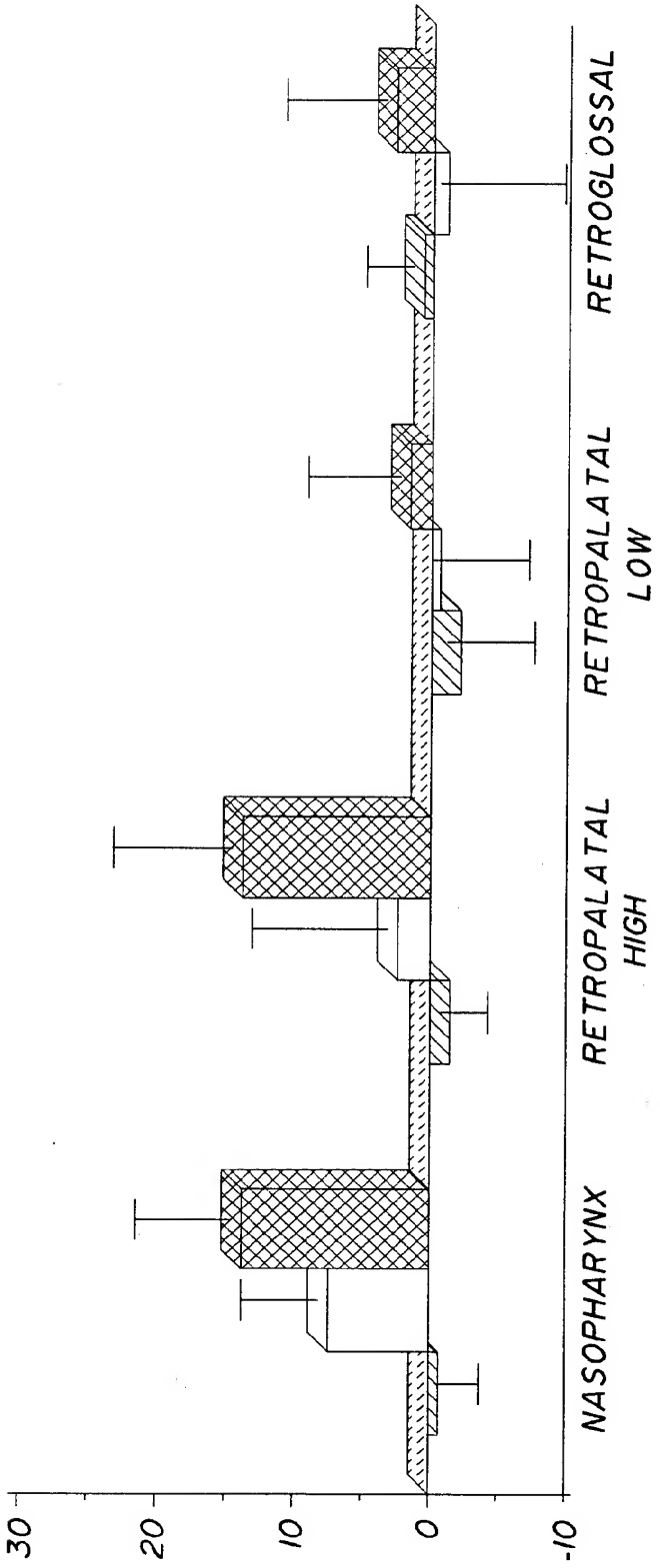
AREA CHANGE IN
EARLY INSPIRATION (mm2)
30
20
10
0
-10
NASOPHARYNX
RETROPALATAL
HIGH
RETROPALATAL
LOW
RETROGLOSSAL

FIG. 7A

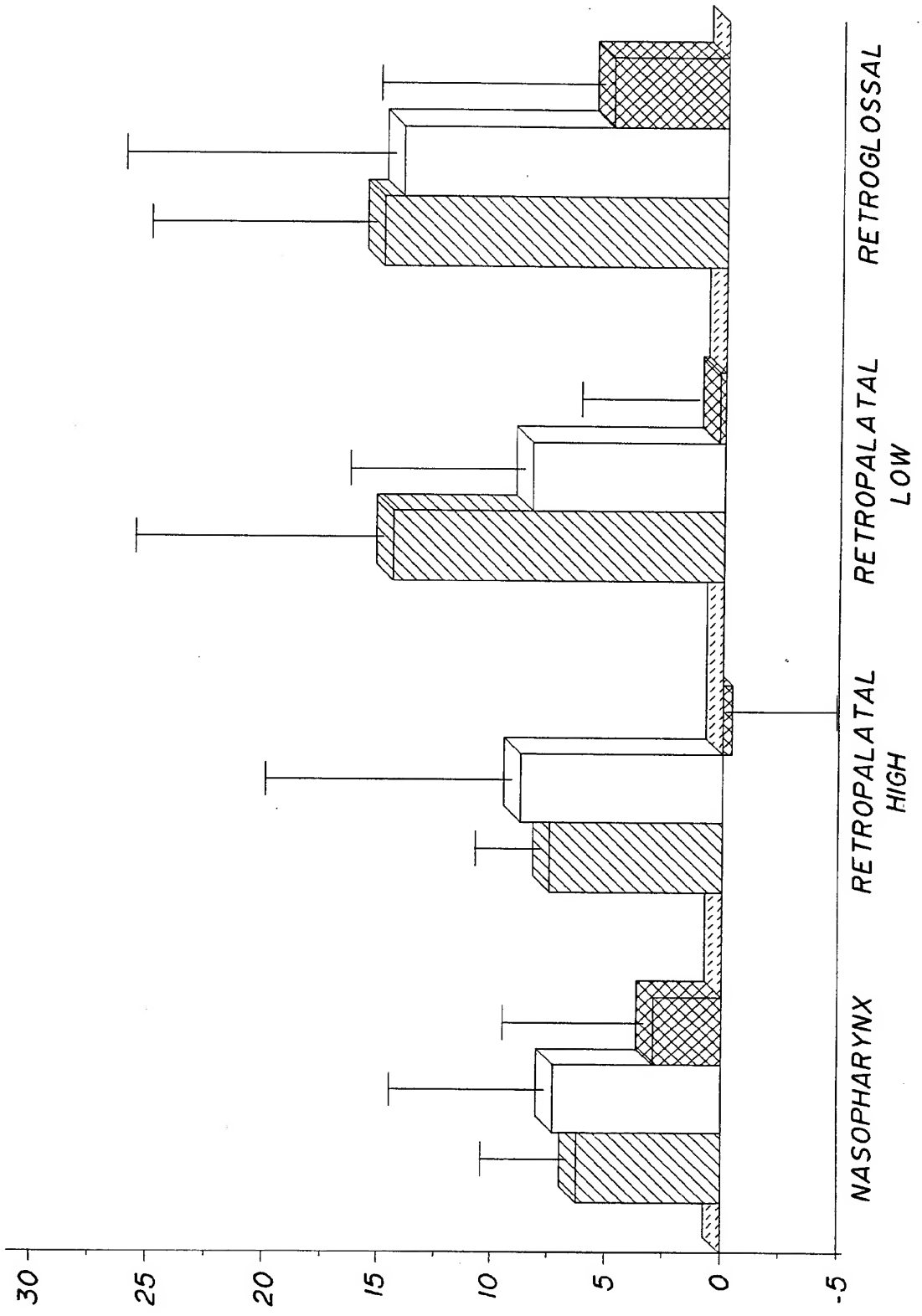
AREA CHANGE IN INSPIRATION (mm²)
30
25
20
15
10
5
0
-5
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL

FIG. 7B

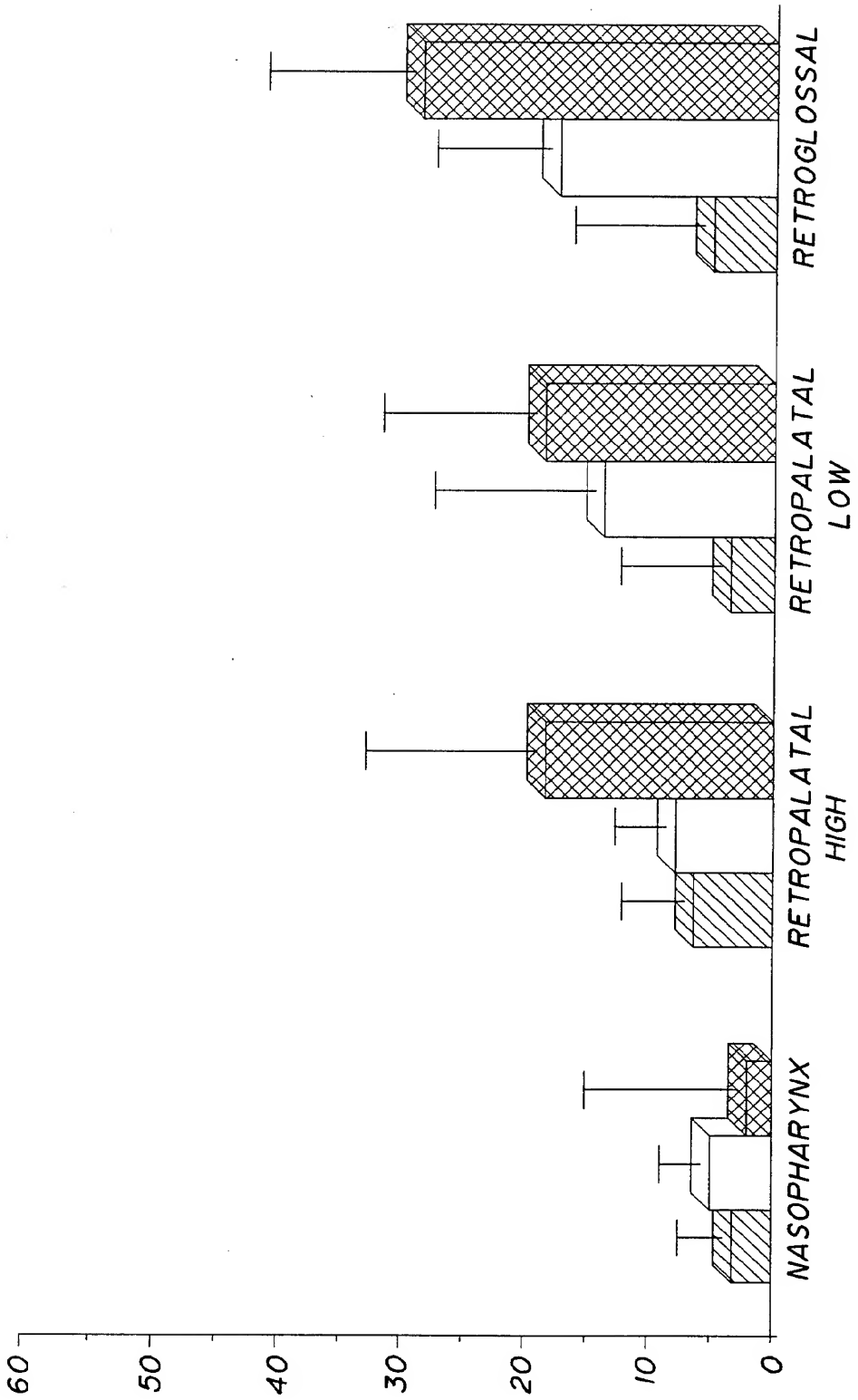
MAXIMUM AREA CHANGE IN EXPIRATION (mm²)
0
10
20
30
40
50
60
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL

FIG. 8A

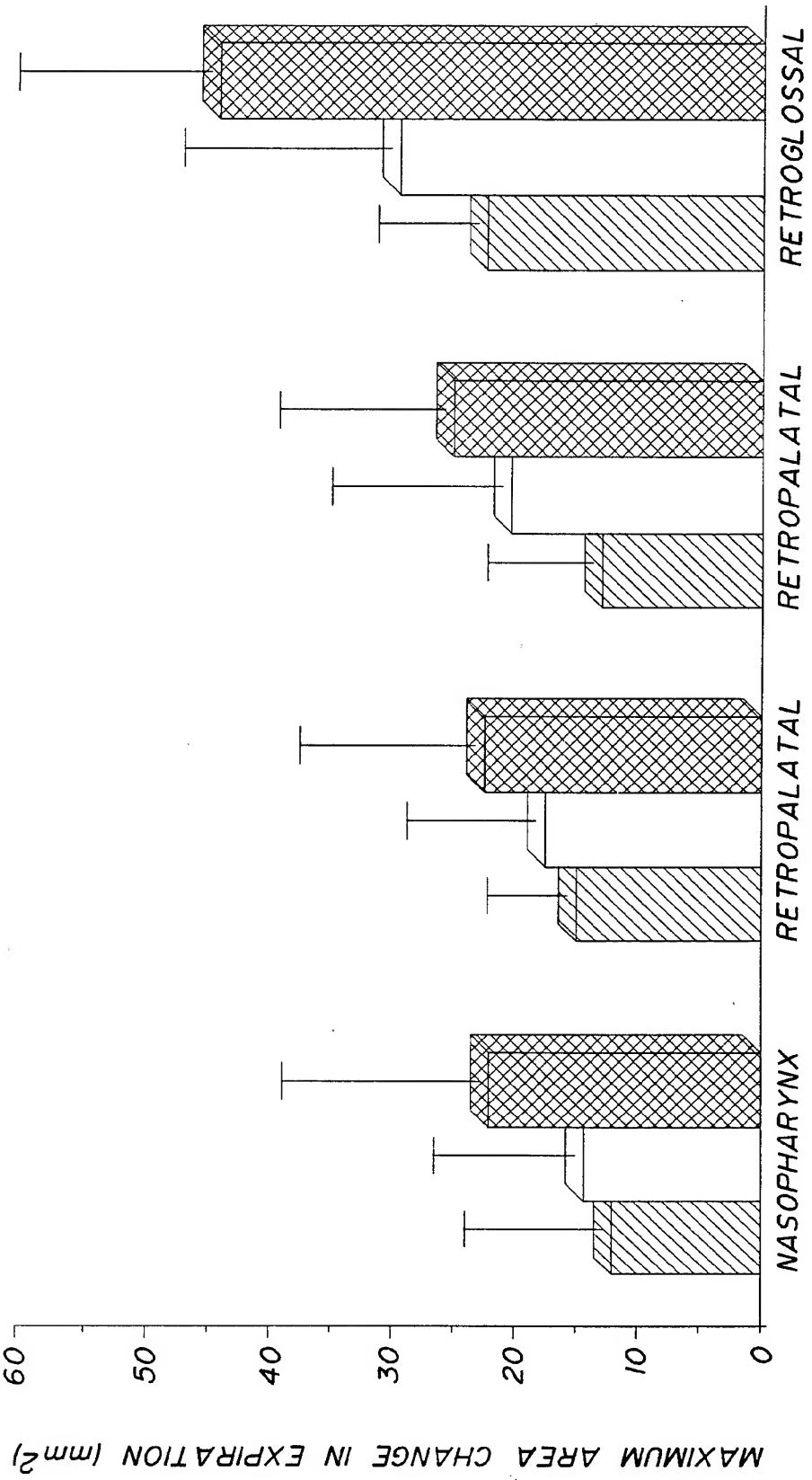
MAXIMUM AREA CHANGE IN EXPIRATION (mm²)
0
10
20
30
40
50
60
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL

FIG. 8B

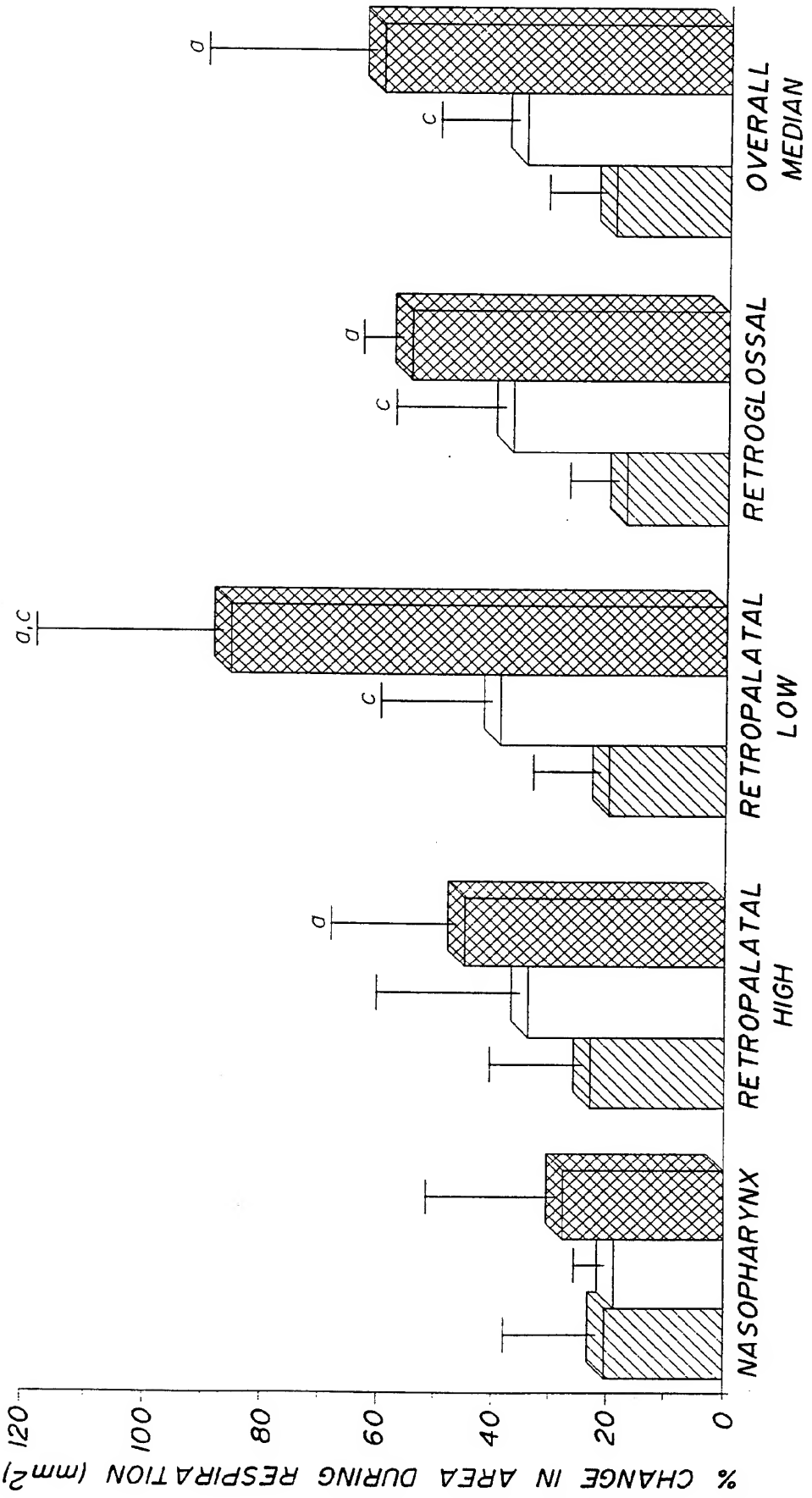
% CHANGE IN AREA DURING RESPIRATION (mm²)
0
20
40
60
80
100
120
NASOPHARYNX
RETROPALATAL HIGH
RETROPALATAL LOW
RETROGLOSSAL
OVERALL MEDIAN
a
c
a,c

FIG. 9

RESPIRATORY CHANGES IN UPPER AIRWAY DIMENSIONS

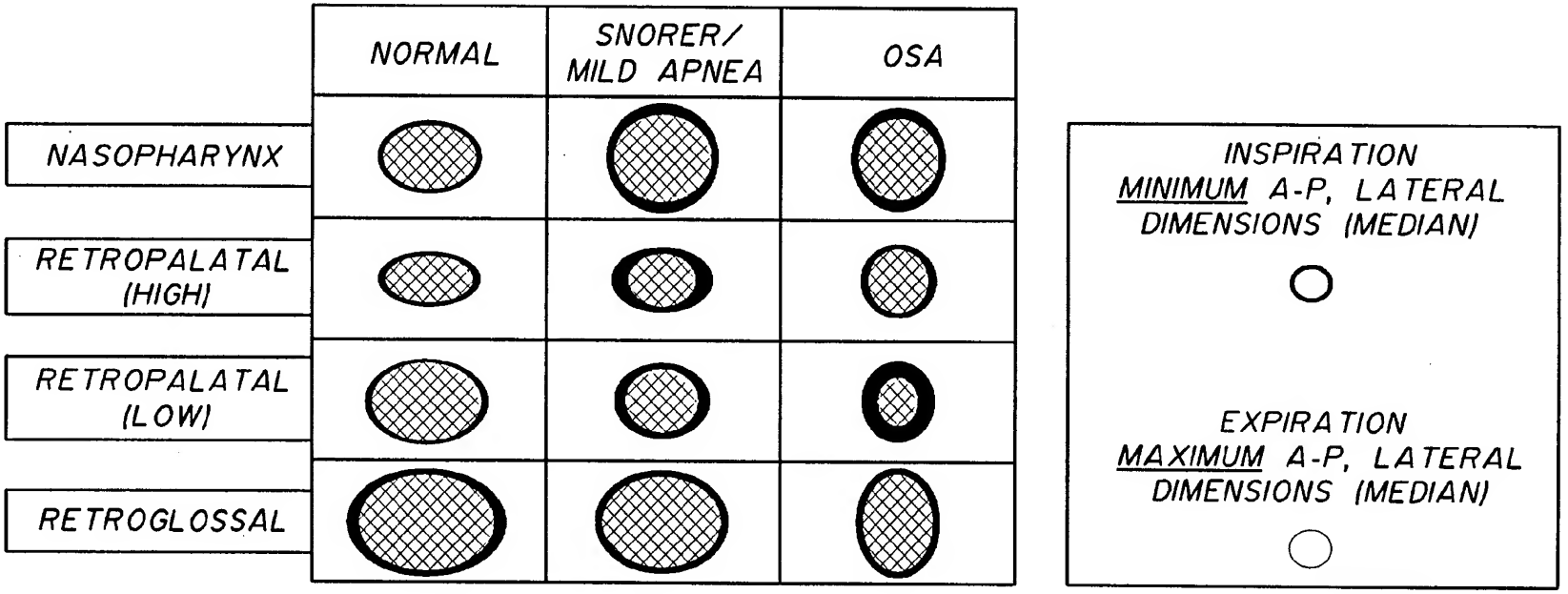

FIG. 10

INTERNATIONAL SEARCH REPORT

International application No.
PCT/US94/03635

A. CLASSIFICATION OF SUBJECT MATTER
IPC(5) :A61M 16/00; A62B 7/00, 18/02; F16K 31/02;
US CL :128/204.23
According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

U.S. : 28/204.18, 204.21, 204.23, 204.26, 205.25

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

NONE

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)

NONE

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X	US, A, 5,134,995, (GRUENKE ET AL.), 04 August 1992. See entire document.	1-12

☐ Further documents are listed in the continuation of Box C. ☐ See patent family annex.

*	Special categories of cited documents:	"T"	later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention
"A"	document defining the general state of the art which is not considered to be part of particular relevance	"X"	document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone
"E"	earlier document published on or after the international filing date	"Y"	document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art
"L"	document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)	"&"	document member of the same patent family
"O"	document referring to an oral disclosure, use, exhibition or other means		
"P"	document published prior to the international filing date but later than the priority date claimed		

Date of the actual completion of the international search
10 JULY 1994

Date of mailing of the international search report
15 SEP 1994

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Form PCT/ISA/210 (second sheet)(July 1992)*